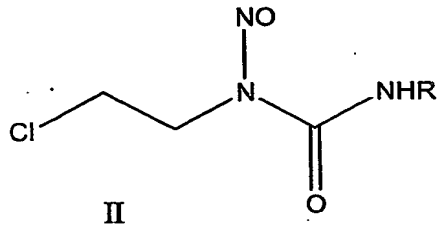
We claim:

1. A formulation comprising an anticancer agent and a base excision repair (BER) inhibitor admixed with pharmaceutically acceptable excipient, wherein the anticancer agent induces formation of AP sites.

2. The formulation of claim 1, wherein said anticancer agent is selected from a DNA oxidizing agent, ultraviolet radiation, a DNA intercalating agent, a radiosensitizing agent, a cross-linking agent, and an alkylating agent.

3. The formulation of claim 2, wherein said anticancer agent is a cross-linking agent.

4. The formulation of claim 3, wherein said a cross-linking agent is a mustine having the structure of formula II:



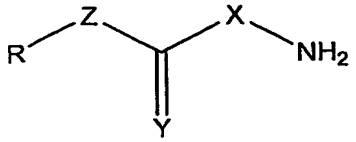
II

wherein R is an optionally substituted hydrocarbon substituent.

5. The formulation of claim 4, wherein said mustine is BCNU.

6. The formulation of any one of claims 2, 4, or 5, wherein said BER inhibitor is an AP endonuclease inhibitor.

7. The formulation of claim 6, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,

Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

8. The formulation of claim 7, wherein said AP endonuclease inhibitor is compound A.

9. The formulation of claim 6, wherein said formulation further comprises a topoisomerase inhibitor.

10. The formulation of claim 9, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

11. The formulation of claim 10, wherein the topoisomerase II inhibitor is etoposide.

12. The formulation of any one of claims 2, 4, or 5, wherein said BER inhibitor is a PARP inhibitor.

13. The formulation of claim 12, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

14. The formulation of claim 13, wherein said formulation further comprises a topoisomerase inhibitor.

15. The formulation of claim 14, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

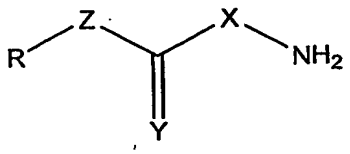
16. The formulation of claim 15, wherein the topoisomerase II inhibitor is etoposide.

17. The formulation of claim 2, wherein said anticancer agent is an alkylating agent.

18. The formulation of claim 17, wherein said alkylating agent is temozolomide.

19. The formulation of any one of claims 17 or 18, wherein said BER inhibitor is an AP endonuclease inhibitor.

20. The formulation of claim 19, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

21. The formulation of claim 20, wherein said AP endonuclease inhibitor is compound A.

22. The formulation of claim 20, wherein said formulation further comprises a topoisomerase inhibitor.

23. The formulation of claim 22, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

24. The formulation of claim 23, wherein the topoisomerase II inhibitor is etoposide.

25. The formulation of claim 2, wherein said anticancer agent is a DNA oxidizing agent.

26. The formulation of claim 25, wherein the DNA oxidizing agent is selected from hydrogen peroxide, bleomycin, and adriamycin.

27. The formulation of claim 2, wherein said anticancer agent is a radiosensitizing agent.

28. The formulation of claim 27, wherein said radiosensitizing agent is IUdR.

29. The formulation of claim 2, wherein said anticancer agent is selected from uracil, hypoxanthine, and 5-FU.

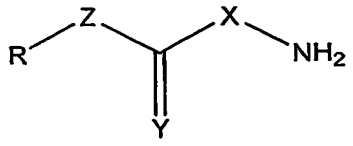
30. The formulation of claim 2, wherein said anticancer agent is ultraviolet radiation.

31. The formulation of claim 30, wherein said ultraviolet radiation is gamma irradiation.

32. The formulation of claim 2, wherein said anticancer agent is selected from temozolomide, IUdR, hydrogen peroxide, bleomycin, adriamycin, uracil, hypoxanthine, and 5-FU.

33. The formulation of claim 32, wherein said BER inhibitor is an AP endonuclease inhibitor.

34. The formulation of claim 33, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

35. The formulation of any one of claims 17 or 18, wherein said BER inhibitor is a PARP inhibitor.

36. The formulation of claim 35, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

37. The formulation of claim 35, wherein said formulation further comprises a topoisomerase inhibitor.

38. The formulation of claim 37, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

39. The formulation of claim 38, wherein the topoisomerase II inhibitor is etoposide.

40. The formulation of claim 1, wherein a dose of the formulation comprises an amount of the anticancer agent that is subtherapeutic when administered in the absence of the base excision repair inhibitor.

41. The formulation of claim 1, wherein said base excision repair inhibitor is selected from an AP endonuclease inhibitor, a DNA glycosylase inhibitor, a DNA polymerase inhibitor, a PARP inhibitor, and a DNA ligase inhibitor.

42. The formulation of claim 41, wherein said base excision repair inhibitor is an AP endonuclease inhibitor.

43. The formulation of claim 41, wherein said base excision repair inhibitor is a PARP inhibitor.

44. The formulation of claim 43, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

45. The formulation of claim 41, wherein said BER inhibitor is an inhibitor of DNA polymerase.

46. The formulation of claim 45, wherein said inhibitor of DNA polymerase inhibits DNA polymerase β, γ, or ε.

47. The formulation of claim 41, wherein said base excision repair inhibitor is a DNA ligase inhibitor.

48. The formulation of claim 47, wherein said DNA ligase inhibitor inhibits the action of DNA ligase I or DNA ligase II.

49. The formulation of any one of claims 7, 10, 13, 16, 20, 24 or 32 which is administered to a patient diagnosed with cancer.

50. The formulation of claim 49, wherein said formulation is administered orally.

51. The formulation of claim 49, wherein said formulation is administered intravenously.

52. The formulation of claim 49, wherein the cancer is selected from carcinomas, melanomas, sarcomas, lymphomas, leukemias, astrocytomas, gliomas, malignant melanomas, chronic lymphocytic leukemia, lung cancers, and breast cancers.

53. The formulation of claim 1, wherein said formulation further comprises a DNA alkyltransferase inhibitor.

54. The formulation of claim 53, wherein said DNA alkyltransferase inhibitor is BG.

55. The formulation of claim 1, wherein said formulation further comprises a topoisomerase inhibitor.

56. The formulation of claim 55, wherein said topoisomerase inhibitor is a topoisomerase I inhibitor.

57. The formulation of claim 55, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

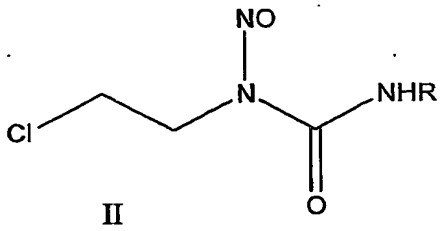
58. The formulation of claim 57, wherein the topoisomerase II inhibitor is etoposide.

59. A method for potentiating a therapeutic effect of an anticancer agent that induces formation of AP sites, comprising administering a base excision repair inhibitor, whereby the base excision repair inhibitor potentiates the effect of the anticancer agent.

60. The method of claim 59, wherein said anticancer agent is selected from a DNA oxidizing agent, ultraviolet radiation, a DNA intercalating agent, a radiosensitizing agent, a cross-linking agent, and an alkylating agent.

61. The method of claim 60, wherein said anticancer agent is a cross-linking agent.

62. The method of claim 61, wherein said a cross-linking agent is a mustine having the structure of formula II:



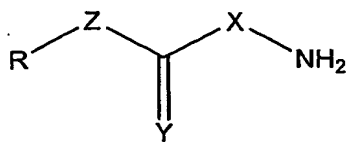
II

wherein R is an optionally substituted hydrocarbon substituent.

63. The method of claim 62, wherein said mustine is BCNU.

64. The method of any one of claims 60, 62, or 63, wherein said BER inhibitor is an AP endonuclease inhibitor.

65. The method of claim 64, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,

Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

66. The method of claim 65, wherein said AP endonuclease inhibitor is compound A.

67. The method of claim 64, wherein said method further comprises administering a topoisomerase inhibitor.

68. The method of claim 67, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

69. The method of claim 68, wherein the topoisomerase II inhibitor is etoposide.

70. The method of any one of claims 60, 62, or 63, wherein said BER inhibitor is a PARP inhibitor.

71. The method of claim 70, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

72. The method of claim 71, wherein said method further comprises administering a topoisomerase inhibitor.

73. The method of claim 72, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

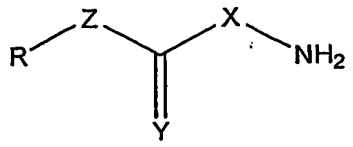
74. The method of claim 73, wherein the topoisomerase II inhibitor is etoposide.

75. The method of claim 60, wherein said anticancer agent is an alkylating agent.

76. The method of claim 75, wherein said alkylating agent is temozolomide.

77. The method of any one of claims 75 or 76, wherein said BER inhibitor is an AP endonuclease inhibitor.

78. The method of claim 77, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

79. The method of claim 78, wherein said AP endonuclease inhibitor is compound A.

80. The method of claim 78, wherein said method further comprises administering a topoisomerase inhibitor.

81. The method of claim 80, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

82. The method of claim 81, wherein the topoisomerase II inhibitor is etoposide.

83. The method of claim 60, wherein said anticancer agent is a DNA oxidizing agent.

84. The method of claim 83, wherein the DNA oxidizing agent is selected from hydrogen peroxide, bleomycin, and adriamycin.

85. The method of claim 60, wherein said anticancer agent is a radiosensitizing agent.

86. The method of claim 85, wherein said radiosensitizing agent is IUdR.

87. The method of claim 60, wherein said anticancer agent is selected from uracil, hypoxanthine, and 5-FU.

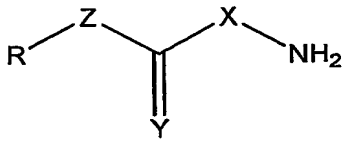
88. The method of claim 60, wherein said anticancer agent is ultraviolet radiation.

89. The method of claim 88, wherein said ultraviolet radiation is gamma irradiation.

90. The method of claim 60, wherein said anticancer agent is selected from temozolomide, IUdR, hydrogen peroxide, bleomycin, adriamycin, uracil, hypoxanthine, and 5-FU.

91. The method of claim 90, wherein said BER inhibitor is an AP endonuclease inhibitor.

92. The method of claim 91, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

93. The method of any one of claims 75 or 76, wherein said BER inhibitor is a PARP inhibitor.

94. The method of claim 93, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

95. The method of claim 93, wherein said method further comprises administering a topoisomerase inhibitor.

96. The method of claim 95, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

97. The method of claim 96, wherein the topoisomerase II inhibitor is etoposide.

98. The method of claim 59, wherein the amount of anticancer agent is subtherapeutic when administered in the absence of the base excision repair inhibitor.

99. The method of claim 59, wherein said base excision repair inhibitor is selected from an AP endonuclease inhibitor, a DNA glycosylase inhibitor, a DNA polymerase inhibitor, a PARP inhibitor, and a DNA ligase inhibitor.

100. The method of claim 99, wherein said base excision repair inhibitor is an AP endonuclease inhibitor.

101. The method of claim 99, wherein said base excision repair inhibitor is a PARP inhibitor.

102. The method of claim 101, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

103. The method of claim 99, wherein said BER inhibitor is an inhibitor of DNA polymerase.

104. The method of claim 103, wherein said inhibitor of DNA polymerase inhibits DNA polymerase β, γ, or ε.

105. The method of claim 99, wherein said base excision repair inhibitor is a DNA ligase inhibitor.

106. The method of claim 105, wherein said DNA ligase inhibitor inhibits the action of DNA ligase I or DNA ligase II.

107. The method of any one of claims 65, 68, 71, 74, 78, 82 or 90, wherein the patient is diagnosed with cancer.

108. The method of claim 107, wherein said base excision repair inhibitor is administered orally.

109. The method of claim 107, wherein said base excision repair inhibitor is administered intravenously.

110. The method of claim 107, wherein the cancer is selected from carcinomas, melanomas, sarcomas, lymphomas, leukemias, astrocytomas, gliomas, malignant melanomas, chronic lymphocytic leukemia, lung cancers, and breast cancers.

111. The method of claim 59, wherein said method further comprises administering a DNA alkyltransferase inhibitor.

112. The method of claim 111, wherein said DNA alkyltransferase inhibitor is BG.

113. The method of claim 59, wherein said method further comprises administering a topoisomerase inhibitor.

114. The method of claim 113, wherein said topoisomerase inhibitor is a topoisomerase I inhibitor.

115. The method of claim 113, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

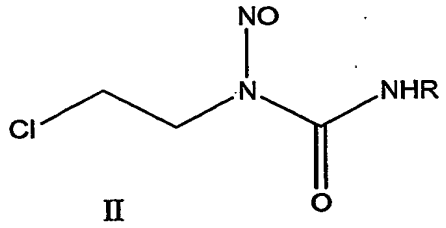
116. The method of claim 115, wherein the topoisomerase II inhibitor is etoposide.

117. A kit comprising a first pharmaceutical preparation comprising an anticancer agent that induces formation of AP sites, a second pharmaceutical preparation comprising a base excision repair inhibitor, and instructions for administering the first and second pharmaceutical preparations to a patient for the treatment of cancer.

118. The kit of claim 117, wherein said anticancer agent is selected from a DNA oxidizing agent, ultraviolet radiation, a DNA intercalating agent, a radiosensitizing agent, a cross-linking agent, and an alkylating agent.

119. The kit of claim 118, wherein said anticancer agent is a cross-linking agent.

120. The kit of claim 119, wherein said a cross-linking agent is a mustine having the structure of formula II:



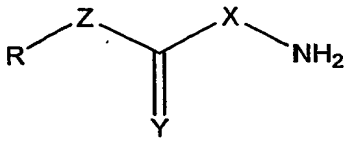
II

wherein R is an optionally substituted hydrocarbon substituent.

121. The kit of claim 120, wherein said mustine is BCNU.

122. The kit of any one of claims 118, 120, or 121, wherein said BER inhibitor is an AP endonuclease inhibitor.

123. The kit of claim 122, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,

Y is O, S, or NH,

Z is absent or represents O, S, or NH, and

R represents a hydrogen or a hydrocarbon moiety,

and pharmaceutically acceptable salts thereof.

124. The kit of claim 123, wherein said AP endonuclease inhibitor is compound A.

125. The kit of claim 122, wherein said kit further comprises a topoisomerase inhibitor.

126. The kit of claim 125, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

127. The kit of claim 126, wherein the topoisomerase II inhibitor is etoposide.

128. The kit of any one of claims 118, 120, or 121, wherein said BER inhibitor is a PARP inhibitor.

129. The kit of claim 128, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

130. The kit of claim 129, wherein said kit further comprises a topoisomerase inhibitor.

131. The kit of claim 130, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

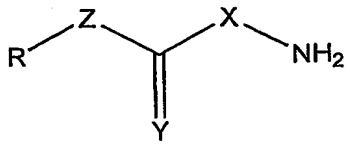
132. The kit of claim 131, wherein the topoisomerase II inhibitor is etoposide.

133. The kit of claim 118, wherein said anticancer agent is an alkylating agent.

134. The kit of claim 133, wherein said alkylating agent is temozolomide.

135. The kit of any one of claims 133 or 134, wherein said BER inhibitor is an AP endonuclease inhibitor.

136. The kit of claim 135, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,

Y is O, S, or NH,

Z is absent or represents O, S, or NH, and

R represents a hydrogen or a hydrocarbon moiety,

and pharmaceutically acceptable salts thereof.

137. The kit of claim 136, wherein said AP endonuclease inhibitor is compound A.

138. The kit of claim 136, wherein said kit further comprises a topoisomerase inhibitor.

139. The kit of claim 138, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

140. The kit of claim 139, wherein the topoisomerase II inhibitor is etoposide.

141. The kit of claim 118, wherein said anticancer agent is a DNA oxidizing agent.

142. The kit of claim 141, wherein the DNA oxidizing agent is selected from hydrogen peroxide, bleomycin, and adriamycin.

143. The kit of claim 118, wherein said anticancer agent is a radiosensitizing agent.

144. The kit of claim 143, wherein said radiosensitizing agent is IUdR.

145. The kit of claim 118, wherein said anticancer agent is selected from uracil, hypoxanthine, and 5-FU.

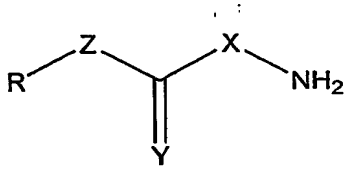
146. The kit of claim 118, wherein said anticancer agent is ultraviolet radiation.

147. The kit of claim 146, wherein said ultraviolet radiation is gamma irradiation.

148. The kit of claim 118, wherein said anticancer agent is selected from temozolomide, IUdR, hydrogen peroxide, bleomycin, adriamycin, uracil, hypoxanthine, and 5-FU.

149. The kit of claim 148, wherein said BER inhibitor is an AP endonuclease inhibitor.

150. The kit of claim 149, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety.
and pharmaceutically acceptable salts thereof.

151. The kit of any one of claims 133 or 134, wherein said BER inhibitor is a PARP inhibitor.

152. The kit of claim 151, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

153. The kit of claim 151, wherein said kit further comprises a topoisomerase inhibitor.

154. The kit of claim 153, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

155. The kit of claim 154, wherein the topoisomerase II inhibitor is etoposide.

156. The kit of claim 117, wherein the amount of the anticancer agent is subtherapeutic when administered in the absence of the base excision repair inhibitor.

157. The kit of claim 117, wherein said base excision repair inhibitor is selected from an AP endonuclease inhibitor, a DNA glycosylase inhibitor, a DNA polymerase inhibitor, a PARP inhibitor, and a DNA ligase inhibitor.

158. The kit of claim 157, wherein said base excision repair inhibitor is an AP endonuclease inhibitor.

159. The kit of claim 157, wherein said base excision repair inhibitor is a PARP inhibitor.

160. The kit of claim 159, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

161. The kit of claim 157, wherein said BER inhibitor is an inhibitor of DNA polymerase.

162. The kit of claim 161, wherein said inhibitor of DNA polymerase inhibits DNA polymerase β, γ, or ε.

163. The kit of claim 157, wherein said base excision repair inhibitor is a DNA ligase inhibitor.

164. The kit of claim 163, wherein said DNA ligase inhibitor inhibits the action of DNA ligase I or DNA ligase II.

165. The kit of any one of claims 117, 123, 126, 129, 132, 136, 140 or 148, wherein the cancer is selected from carcinomas, melanomas, sarcomas, lymphomas, leukemias, astrocytomas, gliomas, malignant melanomas, chronic lymphocytic leukemia, lung cancers, and breast cancers.

166. The kit of claim 117, wherein said kit further comprises a DNA alkyltransferase inhibitor.

167. The kit of claim 166, wherein said DNA alkyltransferase inhibitor is BG.

168. The kit of claim 117, wherein said kit further comprises a topoisomerase inhibitor.

169. The kit of claim 168, wherein said topoisomerase inhibitor is a topoisomerase I inhibitor.

170. The kit of claim 168, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

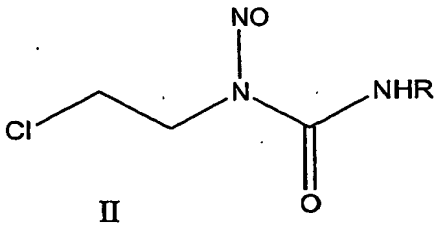
171. The kit of claim 170, wherein the topoisomerase II inhibitor is etoposide.

172. A kit comprising a pharmaceutical preparation comprising a base excision repair inhibitor and instructions for coadministration of the pharmaceutical preparation with an anticancer agent that induces formation of AP sites.

173. The kit of claim 172, wherein said anticancer agent is selected from a DNA oxidizing agent, ultraviolet radiation, a DNA intercalating agent, a radiosensitizing agent, a cross-linking agent, and an alkylating agent.

174. The kit of claim 173, wherein said anticancer agent is a cross-linking agent.

175. The kit of claim 174, wherein said a cross-linking agent is a mustine having the structure of formula II:



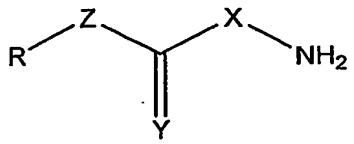
II

wherein R is an optionally substituted hydrocarbon substituent.

176. The kit of claim 175, wherein said mustine is BCNU.

177. The kit of any one of claims 173, 175, or 176, wherein said BER inhibitor is an AP endonuclease inhibitor.

178. The kit of claim 177, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

179. The kit of claim 178, wherein said AP endonuclease inhibitor is compound A.

180. The kit of claim 179, wherein said kit further comprises a topoisomerase inhibitor.

181. The kit of claim 180, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

182. The kit of claim 181, wherein the topoisomerase II inhibitor is etoposide.

183. The kit of any one of claims 173, 175, or 176, wherein said BER inhibitor is a PARP inhibitor.

184. The kit of claim 183, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

185. The kit of claim 184, wherein said kit further comprises a topoisomerase inhibitor.

186. The kit of claim 185, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

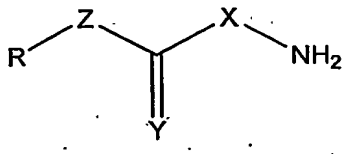
187. The kit of claim 186, wherein the topoisomerase II inhibitor is etoposide.

188. The kit of claim 173, wherein said anticancer agent is an alkylating agent.

189. The kit of claim 188, wherein said alkylating agent is temozolomide.

190. The kit of any one of claims 188 or 189, wherein said BER inhibitor is an AP endonuclease inhibitor.

191. The kit of claim 190, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,
Y is O, S, or NH,
Z is absent or represents O, S, or NH, and
R represents a hydrogen or a hydrocarbon moiety,
and pharmaceutically acceptable salts thereof.

192. The kit of claim 191, wherein said AP endonuclease inhibitor is compound A.

193. The kit of claim 191, wherein said kit further comprises a topoisomerase inhibitor.

194. The kit of claim 193, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

195. The kit of claim 194, wherein the topoisomerase II inhibitor is etoposide.

196. The kit of claim 173, wherein said anticancer agent is a DNA oxidizing agent.

197. The kit of claim 196, wherein the DNA oxidizing agent is selected from hydrogen peroxide, bleomycin, and adriamycin.

198. The kit of claim 173, wherein said anticancer agent is a radiosensitizing agent.

199. The kit of claim 198, wherein said radiosensitizing agent is IUdR.

200. The kit of claim 173, wherein said anticancer agent is selected from uracil, hypoxanthine, and 5-FU.

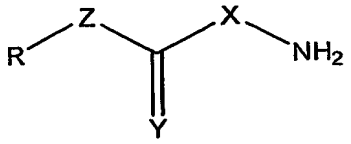
201. The kit of claim 173, wherein said anticancer agent is ultraviolet radiation.

202. The kit of claim 201, wherein said ultraviolet radiation is gamma irradiation.

203. The kit of claim 173, wherein said anticancer agent is selected from temozolomide, IUdR, hydrogen peroxide, bleomycin, adriamycin, uracil, hypoxanthine, and 5-FU.

204. The kit of claim 203, wherein said BER inhibitor is an AP endonuclease inhibitor.

205. The kit of claim 204, wherein said AP endonuclease inhibitor is selected from methoxyamine and a compound having a structure of Formula I:



Formula I

wherein X is O or NH,

Y is O, S, or NH,

Z is absent or represents O, S, or NH, and

R represents a hydrogen or a hydrocarbon moiety,

and pharmaceutically acceptable salts thereof.

206. The kit of any one of claims 188 or 189, wherein said BER inhibitor is a PARP inhibitor.

207. The kit of claim 206, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

208. The kit of claim 206, wherein said kit further comprises a topoisomerase inhibitor.

209. The kit of claim 208, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

210. The kit of claim 212, wherein the topoisomerase II inhibitor is etoposide.

211. The kit of claim 172, wherein the amount of the anticancer agent is subtherapeutic when administered in the absence of the base excision repair inhibitor.

212. The kit of claim 172, wherein said base excision repair inhibitor is selected from an AP endonuclease inhibitor, a DNA glycosylase inhibitor, a DNA polymerase inhibitor, a PARP inhibitor, and a DNA ligase inhibitor.

213. The kit of claim 212, wherein said base excision repair inhibitor is an AP endonuclease inhibitor.

214. The kit of claim 212, wherein said base excision repair inhibitor is a PARP inhibitor.

215. The kit of claim 214, wherein said PARP inhibitor is selected from PD128763, 3-AB, and 6-AN.

216. The kit of claim 212, wherein said BER inhibitor is an inhibitor of DNA polymerase.

217. The kit of claim 216, wherein said inhibitor of DNA polymerase inhibits DNA polymerase β, γ, or ε.

218. The kit of claim 212, wherein said base excision repair inhibitor is a DNA ligase inhibitor.

219. The kit of claim 218, wherein said DNA ligase inhibitor inhibits the action of DNA ligase I or DNA ligase II.

220. The kit of any one of claims 178, 181, 184, 187, 191, 195 or 203, wherein the instructions are for administering the kit to a patient diagnosed with cancer.

221. The kit of claim 220, wherein the instructions are for administering the pharmaceutical preparation orally.

222. The kit of claim 220, wherein the instructions are for administering the pharmaceutical preparation intravenously.

223. The kit of claim 220, wherein the cancer is selected from carcinomas, melanomas, sarcomas, lymphomas, leukemias, astrocytomas, gliomas, malignant melanomas, chronic lymphocytic leukemia, lung cancers, and breast cancers.

224. The kit of claim 172, wherein said kit further comprises a DNA alkyltransferase inhibitor.

225. The kit of claim 224, wherein said DNA alkyltransferase inhibitor is BG.

226. The kit of claim 172, wherein said kit further comprises a topoisomerase inhibitor.

227. The kit of claim 226, wherein said topoisomerase inhibitor is a topoisomerase I inhibitor.

228. The kit of claim 226, wherein said topoisomerase inhibitor is a topoisomerase II inhibitor.

229. The kit of claim 228, wherein the topoisomerase II inhibitor is etoposide.